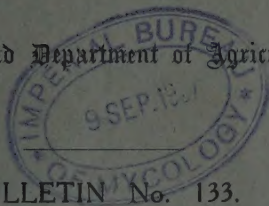


1927

[Issue, July, 1927.]

New Zealand Department of Agriculture.



BULLETIN No. 133.

DRY-ROT OF SWEDES AND TURNIPS: ITS CAUSE AND CONTROL.

BY

G. H. CUNNINGHAM,

Mycologist, Biological Laboratory, Wellington.



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W. A. G. SKINNER, GOVERNMENT PRINTER, WELLINGTON.

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FIG. 1. LESION IN SWEDE-BULB PRODUCED BY THE DRY-ROT ORGANISM. $\times \frac{1}{2}$.

[Photo by E. Bruce Levy.]

FOREWORD.

IN the earlier days of farming in New Zealand an adequate supplementary winter-feed provision could be cheaply and easily provided with the swede crop. Its adaptability to differing soils, high feeding-quality, easy method of utilization, and cheapness of production naturally made it the main winter root crop grown by farmers. To-day the position is very different; disease and insect pests have converted the swede from the most reliable to the most unreliable crop that the farmer can produce. In the drier districts aphids and diamond-back moth largely prohibit its cultivation, and in the wetter regions club-root and particularly dry-rot are so disastrous in their action that the farmer has either to abandon swede cultivation, or has to be content with continuing growing a crop which he is never sure will be successful and available for the purpose for which it is really required—namely, mid-winter and early spring feeding.

The ability to grow satisfactory swede crops means vastly increasing the winter carrying-capacity of any holding, with subsequent better utilization of seasonal grass-production. For this reason the adequate control and elimination, if possible, of swede-diseases is of the utmost economic importance. It is difficult to hazard in cold figures what the elimination of dry-rot would mean to New Zealand farming. Suffice it to say that over a large part of New Zealand it would make farm-management far easier, and appreciably reduce the cost of production of animal products. As animal products represent the basal selling crops of the New Zealand farmer, any reduction in their costs of production, derived by methods other than reduction in land and labour charges, will be reflected in improved conditions to all.

Such being the case, the results of the long series of investigations on dry-rot in swedes and turnips, carried out by the Biological Laboratory and brought by the brilliant work of Dr. Cunningham to successful completion, are of far-reaching importance.

So far as practical mycology is concerned, research can be viewed as merely a means to an end, and with regard to field crops, at any rate, that end should be the total elimination of whatever disease the research is directed against. In the majority of cases in the past, however, economic research against crop-diseases has set up as its goal the formulation of methods—often quite impracticable from the general farm-management standpoint—that tend to reduce the effects of diseases, rather than methods which will eliminate them. If a certain method reduces the loss from 50 per cent. to 5 per cent., research claims a notable victory against plant-disease; but in point of fact such method has no relation whatever to elimination, and it is the stamping-out rather than the control of field-crop diseases that has any real significance.

To my mind the real practical value of this bulletin lies in the fact that careful research on the cause and distribution of dry-rot has led to results that make it quite feasible to stamp out dry-rot from New Zealand, and, for the matter of that, from any country where the swede is grown and the disease is present.

It has been conclusively proved that dry-rot is carried in the seed, and if the seed sown is disease-free, or the disease in the seeds has been killed, the resultant crop will be disease-free, provided outside infection does not take place. Outside infection can take place only when pieces of diseased roots from a crop grown on the same ground in the previous year are present, or when spores are introduced from a crop sown with seed containing the disease in a living form. The first-named means of infection can easily be guarded against by the farmer, and if nothing but disease-free seed is sown the second means will become inoperative.

The total elimination of the disease thus rests entirely on nothing but disease-free swede and turnip seed being sown by the farmer, and a workable method whereby this is secured should eliminate dry-rot from New Zealand within a single season. The treatment of disease-infected seed by the farmer himself is not feasible, and even if it were so it would never entirely be put into practice. But the technique and methods fully described in the bulletin should make it perfectly simple for turnip-seed and swede-seed growers, be they in New Zealand or overseas, to supply nothing but disease-free seed, and in this way one of our most serious of all field-crop diseases could be completely stamped out of the Dominion.

A. H. COCKAYNE,
Director, Fields Division.

Wellington, June, 1927.

TABLE OF CONTENTS.

	Page
Foreword, by A. H. Cockayne	3
Introduction	7
Historical	7
Dissemination of the Disease	8
Experiments to determine whether the Disease was soil-borne ..	8
Experiments to determine whether the Disease was seed-borne ..	10
Methods used	10
Results	12
Experiments to determine the Part of the Seed in which the Disease is present	12
Life-history of the Causal Organism	13
Method by which it spreads in the Field	15
Manner in which the Seed becomes infected	20
Cultural Studies of the Causal Organism	20
Inoculation Experiments	20
With New Zealand Strains	22
With Foreign Strains	22
Comparative Cultural Experiments	23
Description of Strains	25
Morphological Studies of the Causal Organism	36
Description of the Causal Organism	36
Synonymy	38
Common Names	38
Hosts	39
Symptomatology	39
On Leaves	40
On Stems	40
On Siliquas	40
On Seeds	40
On Bulbs	40
Varietal Resistance	41
Development of the Pycnidia	41
Laboratory Experiments in Control	43
Chemical Treatments	43
I. Mercuric Chloride	43
II. Mercurio-phenol Compounds	44
Hot-water Treatments	44
Presoak plus Ten Minutes' Dip	44
Dip without Presoak	45
Hot Semesan Treatments	45
Field Experiments in Control	46
"Mother-seed" Method	48
Summary	49
Literature cited	51

DRY-ROT OF SWEDES AND TURNIPS: ITS CAUSE AND CONTROL

INTRODUCTION.

SINCE 1905, when it was first recorded in New Zealand, dry-rot has become the most serious disease of the swede. In a season favourable to its spread it may destroy from 50 to 100 per cent. of the crop. On the turnip its effects are generally less marked, but even here the loss may amount to 40 per cent. Infected bulbs become a total loss, for they quickly rot and become worthless for fodder.

The economic effects of this disease are far-reaching, for the swede is the most valuable of all fodders for carrying stock over the winter months, and so severe have been the losses through dry-rot that farmers have been forced to substitute other and inferior crops for the purpose. The problem of its control is becoming more urgent in view of the increasing practice of pasture top-dressing, since the consequent rush of summer feed requires more winter feed for its proper utilization. Further, farmers are loath to break up their rejuvenated pastures in order to get "clean" land each year for swede-growing—for it is a common belief that heavy losses follow where swedes are grown twice in succession on the same land.

HISTORICAL.

Dry-rot was first recorded for New Zealand in 1905 by Mr. T. W. Kirk (5), from specimens forwarded from Portobello, Otago. He identified the causal organism as *Phoma Napobrassicae* Rostr., and this name has been used by subsequent New Zealand workers. Kirk believed the disease to be carried over from year to year by means of decayed bulbs left in the ground. In 1907 (6) he stated a large area of turnips had been ruined by dry-rot in both North and South Islands.

No further reference to this disease was published until 1918, when Mr. A. H. Cockayne (2) made a survey of the position and suggested methods of control.

In 1918 Mr. E. Bruce Levy commenced a series of field investigations on the control of dry-rot—investigations which were continued until 1922. His first report (7) dealt with three series of experiments—(a) manurial tests, (b) varietal trials, and (c) seed-origin trials. He claimed that manuring tended to a better crop, but that manured plants were more subject to infection than were non-manured. Varietal trials were unsatisfactory, all those varieties tried being subject to infection, the least susceptible showing 73 per cent. of infected bulbs. Nor did the seed-origin trials prove to be of value save to bring to light the fact that there was considerable deviation from the varietal type in the same variety as supplied by different seedsmen. He dealt with this matter more fully in a later paper (8).

Levy's second report (9) dealt with a series of experiments calculated with a view to (a) producing a long-growing, slowly

maturing bulb; (*b*) producing a large number of small, closely growing, hardy bulbs; (*c*) ascertaining the effects of external chemical seed-sterilization and spraying on the crop; (*d*) studying the crop development following different times of sowing; (*e*) ascertaining the effect of using seed raised in different localities and of varying origin; (*f*) studying closely allied species of plants that may act as hosts for the dry-rot organism; (*g*) ascertaining effects of varietal trials in relative immunity; (*h*) selecting by trial disease-resistant varieties.

No factors of value from the control viewpoint were brought to light in this series of experiments, but his field observations on the spread of dry-rot led Levy to believe there were three stages of infection, which he termed (i) seedling infection, (ii) primary infection, and (iii) secondary infection. He came to the conclusion that the disease overwintered in the soil. Consequently, in his third (final) report (10), Levy dealt with a series of experiments carried out with a view to destroying this soil-borne contagium. In these experiments he used burnt lime, sulphur, copper and iron sulphate, formalin solution, carbon bisulphide, and bordeaux mixture 4-6-40. He found that it was not possible to control the disease by this means, and concluded that the only feasible method lay in crop rotation—a seven-year rotation being suggested.

In other countries dry-rot of swedes and turnips has apparently been of little moment, so few references regarding it having been published, these mostly being in the form of mere references in annual reports, plant-disease surveys, and the like. As will be shown subsequently, the organism causing dry-rot is a serious disease of Brassicas in Europe and North America. The position, then, when the writer commenced work on the dry-rot problem in October, 1924, was that the disease was considered to be soil-borne: consequently, in order to deal satisfactorily with the disease he had first to ascertain the manner in which the disease was disseminated, the morphology of the causal organism (in order to determine its specific identity), its life-history, and from this material work out, if possible, a satisfactory method of control.

DISSEMINATION.

Before attempts at control measures of dry-rot could be worked out it was necessary to determine the means of dissemination of the causal organism—*e.g.*, to find out the manner in which infection occurred each season. Two possibilities were open: either (*a*) that the disease was carried over in the soil from season to season (soil-borne), or (*b*) that the disease was carried in or on the seed (seed-borne).

(a) Experiments to determine whether the Disease was soil-borne.

The disease may have overwintered in the soil by (i) spores, (ii) vegetative mycelium in the soil or in vegetable matter, (iii) by means of (i) or (ii) in sheep-droppings.

Experiments (detailed later) showed that free spores were viable for but a short time—from five to ten days—therefore it was considered dry-rot was not carried in the soil by means of spores.

To ascertain whether it could persist by means of mycelium, a series of cultures was made from soil taken from the Gore Experimental

Area, part of which had been in swedes shortly before the samples were taken (some two months before), part in swedes the previous year, and part in swedes two years previously. Samples were taken from low-lying wet areas, dry hilltops, strips variously manured, from gateways near paddocks used as a run-off for sheep fed on swedes which had been severely attacked with dry-rot the previous season, and vegetable debris was secured wherever possible from these areas, for it was considered possible that the organism may remain as a saprophyte in this class of tissue. In all some seventy samples were secured—two samples being taken from each locality, one from on or near the surface, the other 3 in. deep; and four platings were made (on to culture media) from each sample, making a total of 280 cultures in all.

From every culture several fungi were secured, but in no case was the dry-rot organism—nor, in fact, any *Phoma*—obtained, so that it was considered the organism could not live over in soil, even that which had just been in swedes.

Then an attempt was made to find whether the organism could live in decaying vegetable debris. Numerous cultures were made from tissues partially buried in the ground (taken from different parts of fields which had been in swedes prior to the experiment and one year previously).

Soil-samples were washed, vegetable debris picked out and plated. No *Phoma* was secured in these cultures, though again other fungi were abundant. Next, bulbs which had been attacked by the fungus and were dried out ("mummied") were secured, these being obtained from corners of the paddock, small unploughed gullies, &c. These all came from areas which had but recently been in swedes; for, although careful search was made in areas which had been in swedes the previous year, no mummied bulbs were secured. Cultures made from these frequently yielded the dry-rot organism. Sections of these mummied bulbs showed that the fungus was able to overwinter either in the form of chlamydospores or pycnidia containing viable spores.

Thus this cultural evidence showed that the organism was not present in soil of a paddock which had been in swedes recently fed off, nor was it present in rotting vegetable debris taken from these same areas; but that it was present in a small number of mummied bulbs lying on the surface of the ground.

Field evidence as to its being soil-borne lies in the fact that the disease is frequently more severe on land which has been in swedes for two consecutive seasons. From the previous experiments one can but assume that the disease in such cases spreads from occasional mummied bulbs left lying on the surface of the ground. But these are seldom present unless the crop has been fed off late (August or September) and the second crop sown early (November), for in such cases the mummied bulbs would not have sufficient time to decay, and would then serve as initial centres of infection.

The possibility of the disease being carried by sheep, &c., from paddock to paddock had to be considered. To ascertain whether the disease was carried in dung of sheep the following experiment was undertaken. Fresh droppings, and those which had dried out, from sheep fed almost exclusively on swedes which had been severely attacked with dry-rot were collected. From several, cultures were made directly; part was macerated in water, centrifuged, and the

fibrous residue plated; part was broken up in water, and the larger portions of vegetable debris selected and plated. In all cases fungi were obtained, but in no case was the dry-rot organism observed. As platings made from gateways to sheep run-offs have been recorded as being negative, this evidence shows that sheep are not a factor in dissemination of this disease, even when fed on a severely dry-rotted crop. This may in part be due to the fact that sheep are adverse to eating those portions of swedes which have been infected, part to the organism not being able to withstand passage through the alimentary tract.

From the negative evidence of these experiments, and the fact that dry-rot so frequently appeared on virgin land, the writer was led to assume that dry-rot was not usually soil-borne. Therefore a series of experiments were commenced with a view to determining whether the disease was seed-borne.

(b) Experiments to determine whether the Disease was seed-borne.

One-pound samples were secured of eleven commercial swede-seed lines on the market. A fair sample of each was taken, and twenty lots, each of 100 seeds, were counted out. Each hundred-seed lot was surface-sterilized for ten minutes in acidulated mercuric-chloride

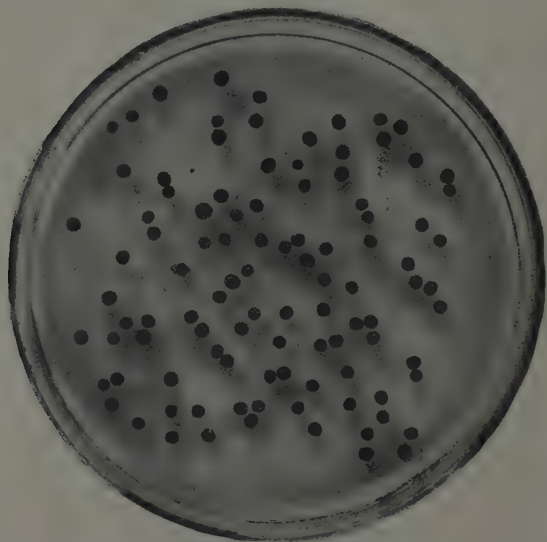


FIG. 2. METHOD OF DETERMINING THE PRESENCE OF THE DRY-ROT ORGANISM IN A SEED LINE: ONE HUNDRED SEEDS PLATED (AFTER SURFACE STERILIZATION) TO CULTURE MEDIA IN PETRIE DISH. NATURAL SIZE.

[Photo by H. Drake.

solution, 1-1,000, washed in distilled water, and plated on to potato dextrose agar, the seed being evenly sprinkled over each plate (Fig. 2). In all twenty plates of each commercial sample were prepared. As a rule almost pure cultures of the dry-rot organism were obtained (Figs. 3, 4), consequently it was relatively a simple matter to

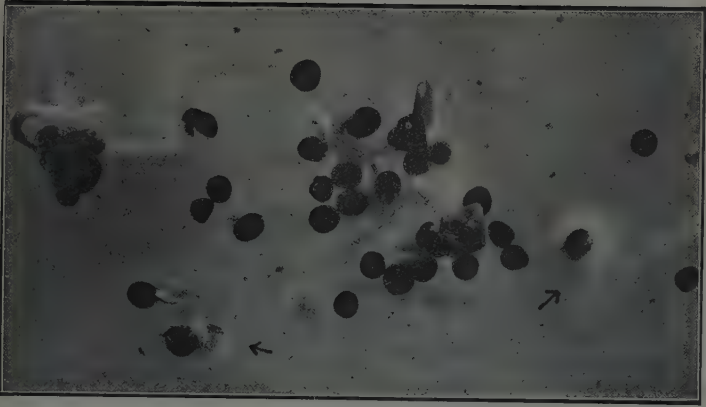


FIG. 3. FUNGUS (INDICATED BY ARROWS) GROWING FROM SEEDS TO MEDIA FIVE DAYS AFTER PLATING. $\times 5$.

[Photo by H. Drake.]



FIG. 4. COLONY OF THE DRY-ROT ORGANISM DEVELOPING FROM SEED SEVEN DAYS AFTER PLATING. SLIGHTLY ENLARGED.

[Photo by H. Drake.]

determine the percentage of disease present, especially at the end of the fifth day, when the colonies were large and clearly defined.

The following table shows the number of colonies obtained from each commercial sample. As the seeds were in hundred lots, this gives also the percentage of disease present in the line, a percentage which over a large number of experiments was found to vary only about 0.1 per cent., provided the seed was taken always from the same sample.

TABLE I.—RESULTS OF PLATING SEED TO MEDIA (1926 Seed).

Variety.	Result.	Variety.	Result.
1	1.0 per cent. infection.	7	0.9 per cent. infection.
2	0.1 per cent. infection.	8	Disease-free.
3	Disease-free.	9	"
4	3.5 per cent. infection.	10	"
5	0.1 per cent. infection.	11	"
6	1.0 per cent. infection.		

This table shows that of eleven commercial lines of 1926 seed, five were disease-free, two had 0.1 per cent. infection, one had 0.9 per cent., two had 1.0 per cent., and one had 3.5 per cent. Each colony was isolated and compared with those obtained from dry-rot lesions on swede bulbs; further, each was inoculated into swede bulbs, and produced the characteristic dry-rot lesion. This proved that the dry-rot organism was carried in the seed, and that this seed-borne organism was capable of producing typical dry-rot lesions in swede bulbs.

The method outlined above has regularly been applied during the past two years to determine the presence (and percentage) or absence of the organism in the seed, and has proved quite reliable.

A second method was attempted when it was found that lesions were found on cotyledons of seedlings. This consisted in sowing on seed-germinator trays (trays of asbestos, covered with sterilized felt, over which were placed sheets of sterile blotting-paper—the whole saturated with distilled water), 2,000 seeds of each sample to be tested, and in examining the resultant seedlings daily. Those showing lesions on cotyledons were removed (Fig. 5), and the cotyledons cut off and plated on media. This method proved unsatisfactory, for it was noted that, although a large proportion of the seedlings showed lesions, only a few of these produced dry-rot colonies. Most lesions were found to be sterile—that is, no pathogen was secured from them, and many were found to be due to a species of *Alternaria*. The dry-rot organism was regularly obtained, but the percentage varied to such an extent as to prove the method unreliable.

Having established the fact that the dry-rot organism was seed-borne, the next point to establish was whether it was carried externally or internally. As the organism was obtained regularly from infected lines, even when the seed was surface-sterilized for ten minutes, it was concluded that it was either in the seed-coat (testa) or in the embryonic cotyledons. Therefore 1,000 seeds from a line containing approximately 4 per cent. of the disease were, after surface-sterilization for ten minutes in acidulated HgCl₂, soaked in distilled water (to soften and swell the

seed) for twenty-four hours and the testa dissected from each. Testas and embryos were then plated separately on to media, with the following results, after five days: Testas, 4 per cent. of the dry-rot organism present; embryos, no dry-rot organism present.

A large number of the embryos developed seedlings, despite this treatment, and 200 of these were removed and replated, but no pathogen was obtained from any of them, despite the fact that in all cases they were laid longitudinally so that one or both cotyledons were in contact with the media.



FIG. 5. GERMINATING SWEDE SEEDLINGS, SHOWING THE MANNER IN WHICH TESTAS ARE CARRIED UPWARDS ON THE COTYLEDONS TIPS. $\times 2$.

Arrow points to dry-rot lesion on cotyledon of plant on the right.

[Photo by H. Drake.

This established the fact that the disease is seed-borne, and that the causal organism is present in the testa but not in the embryo, a matter of considerable importance in working out a control, and also in the cycle of infection, as will be shown.

LIFE-HISTORY OF THE CAUSAL ORGANISM.

The following details have been worked out partly in the laboratory and partly in the field. As will appear, this knowledge has been of great service in enabling a control to be worked out.

As shown, the disease is present in the seed-coat but not in the embryo. When the seed germinates the testa is frequently carried upwards on the tips of the cotyledons (Fig. 5). Ere the latter expand

the fungus grows into the cotyledon-tips, there producing a small lesion on one or both cotyledons (Fig. 6). The lesion is produced by the fungus hyphæ penetrating into and destroying the cells. These turn brown and die, and in them knots of hyphæ are produced, which eventually take the form of flask-shaped receptacles or pycnidia (Fig. 7). In these structures one-celled, colourless spores are produced in abundance. The pycnidium is embedded in the tissues of the leaf, and opens to the exterior by means of a small pore (termed an ostiolum). The spores are embedded in the pycnidium in a mucilaginous matrix, which, being hygroscopic, absorbs moisture and swells. Consequently, in wet or humid weather the mucilage swells to such an extent that it is forced



FIG. 6. SEEDLING LEAF OF SWEDE TWELVE DAYS AFTER GERMINATION, SHOWING LESION PRODUCED BY THE DRY-ROT ORGANISM. $\times 5$.

[Photo by H. Drake.]

through the ostiolum as a small gelatinous tendril, in which are embedded the spores. In this manner spore-discharge occurs, but, as such is possible only in the presence of moisture, discharge can occur only during wet or humid weather. It will thus be seen that no discharge is possible during dry weather. The gelatinous tendril is soluble in water, consequently in wet weather it takes a liquid consistency and is washed down the leaf or spattered in raindrops to leaves of contiguous plants carrying numerous spores. Here again spore-dispersion is dependent on abundant moisture and can occur only if such is present. If a dry spell prevails, the spore-tendril dries out and becomes firmly fixed to the surface of the leaf (or bulb, as the case may be); consequently, until moisture is again present, dispersion of the spores is not possible. Cultural experiments have shown that spores embedded in

this mucilage are capable of remaining viable for over six weeks, but if the mucilage is removed the spores live for a short period—five to ten days, or even less.

Infected seedlings thus are present in the field about two weeks or less after the seed has been sown, their number varying according to the amount of infection present in the seed. If dry conditions are met with after sowing, or if the seed is sown late in the season (say, in January), the probability is that these initial infection sources are considerably lessened, and possibly, in extreme cases, eliminated.



FIG. 7. LESION PRODUCED BY THE DRY-ROT ORGANISM IN SWEDE LEAF. THE BLACK BODIES ARE PYCNIDIA. $\times 10$.

[Photo by H. Draht.

The fungus spreads by means of the spores to other leaves of the same plant, and frequently to other plants in the vicinity. Apart from forming lesions on the leaves it does little damage, however, for as yet no bulbs are formed. Field examination, too, shows at this stage that the disease seldom spreads beyond the initially infected plant, or at most beyond those in its immediate vicinity. Such a condition prevails until from six to eight weeks after sowing, when the bulb is about $\frac{1}{4}$ in. to 1 in. in thickness. At this stage dry-rot becomes noticeable owing to the death of occasional plants in the field, these showing a rapid wilt and discoloration of the leaves. Examination shows the crown of such

plants to be completely rotted (Fig. 8), although the lower part and root of the bulb may be quite healthy. Infection of these individual plants occurs shortly after bulb-formation, spores being washed from



FIG. 8. NECK INFECTION OF SWEDE BULB WITH THE DRY-ROT ORGANISM.

Note the complete destruction of all tissues in this region. Natural size.

[Photo by H. Drake.

the leaves by way of the petioles to the crown. Here penetration of the tissues is effected, decay commences, and in the course of a few days is produced a lesion characteristic of early dry-rot infection. In

this lesion numerous pycnidia are formed, and from these, during moist weather, spore-tendrils are produced. These bulbs serve as the chief means whereby the disease carries over the earlier parts of the season (Fig. 9), for the fungus lives in the tissues for several weeks, and, wherever the weather conditions are moist, during the whole of this period is capable of producing spores. These, as has been shown, are embedded in mucilage. Now, as leaf-motion in the field is constant, leaves frequently brush these tendrils and in turn become infected; or the tendrils may thus be carried on to other surfaces, and spores washed to the crowns of contiguous plants by rain. The economic significance of this primary bulb-infection is that infection centres, capable of infecting over a long period, are established over the field (Fig. 9). From



FIG. 9. PRIMARY BULB INFECTION.

Stakes were placed beside individual bulbs in this row when these only were showing the presence of the disease. Note how contiguous bulbs have since become infected.

[Photo by E. Bruce Levy.]

these centres the disease spreads to contiguous plants in the rows, so that within a few weeks after the scattered plants have become noticeable others in the vicinity show lesions, and in turn rot (Fig. 10).

It should be stressed that infection can occur in the field only when the spores are brought in contact with the leaf or bulb of a plant (Fig. 11). Such contact is effected by means of the leaves, or by the spores (in mucilage) being splashed to other plants in raindrops. And that is the reason why infected plants at this or a later stage occur in the nature of small irregularly circular areas, and not simultaneously over the whole field. Wind plays here only an indirect part in spore-dispersion—that is, by moving the leaves about, or in blowing drops

Inset 1—Dry-rot.



FIG. 10. SHOWING THE IRREGULAR CIRCULAR INFECTION CENTRES ESTABLISHED IN THE FIELD. AREA OUTLINED IN WHITE.

[Photo by E. Bruce Levy.]



FIG. 11. SPREAD OF THE DISEASE FROM DISEASED TO HEALTHY PLANTS.

Initial infection occurred on plant to right (not shown in photo), the disease then spreading to the bulb on the right, and from this to the second on the right.

[Photo by E. Bruce Levy.]

of rain containing spores to contiguous plants. It follows, therefore, that if one field is separated from another by a road or similar gap the disease cannot be transmitted across this gap, provided insects, animals, or cultural implements do not carry the disease from diseased to healthy plants across such gaps. As moisture is so necessary to spore-dispersion, this explains the reason why the disease is more severe in wet seasons or in wet and low-lying areas in the field (Fig. 10); for under these conditions spore-production is continuous, and infection can occur almost continuously. Conversely, dry-weather conditions inhibit infection; so that in certain localities of New Zealand, and in certain seasons throughout, little or no dry-rot is present.

From these small initial infection areas the disease gradually spreads outwards, so that frequently the areas merge and their individuality is lost. Should wet-weather conditions prevail the disease may thus in the course of two or three months spread throughout the whole of the field; in fact, it is usual in the South to find that in a favourable dry-rot season 80 per cent. or even more of the crop may be affected.

As has been shown, initial bulb-infection is invariably followed by death of the plant. This is due doubtless to the fact that the diameter of the bulb is slight, and the fungus quickly destroys all tissues in its vicinity. Later, however, as the bulbs increase in diameter, the number of lesions may become much greater, but each may penetrate to only a slight extent, so that the bulb, although with numerous lesions, may remain sound for many months. Complete decay of these large bulbs is by no means common, save towards the end of the season, when they may become dried out and present a "mummied" appearance. Such bulbs are a possible source of infection the following season, for unless buried they may lie on the ground (even when ploughed, as the cultural implements frequently bring them to the surface) until the next crop has appeared. This applies only when a crop has been fed off late, and the following crop sown early.

Leaf-infection of necessity occurs first in the life-cycle, but later, when the bulbs are formed, bulb-infection is the more common, though at all stages leaf lesions may be found. Lesions develop chiefly on the neck and crown of the bulb, less commonly on the side, save where much exposed, and rarely indeed at or below the ground-level. The explanation of this is that most of the spores are distributed by leaf-contact, first with diseased, then to healthy leaves or bulbs. Spores are therefore washed from leaves down the petioles to the crown, or deposited there by other leaves directly. The rarity of ground-level infection shows that seldom are the spores carried to this part, and this can be explained when the shape of the average bulb is considered. If a bulb is normally upright its shoulder prevents the spores being washed to the lower portion; if, on the other hand, the bulb is inclined, the shoulder is no longer a protection, and heavy infection may result. Infections at or below ground-level are, of course, merely accidental, on account of their rarity: they occur through spores being deposited on the bulb at this point, those below ground-level occurring only when the soil has been forced away from the bulb, such as occurs occasionally when the bulb has been moved by wind or animals.

Manner in which the Seed becomes infected.

In order to ascertain the manner in which the seed becomes infected advantage was taken of the attempts made by Mr. C. W. Body, Kelso, Otago, to grow seed from bulbs. Mr. Body transplanted a number of mature, apparently sound bulbs to a part of a paddock. As the bulbs began to go off with dry-rot others were planted in their stead. Nevertheless, so severely infected did these plants become that few reached the stage of maturing a seed-crop.

Specimens secured from time to time showed that as the flowering-spike developed, the leaves on it became infected with dry-rot, lesions typical of this disease being produced. They appeared also on the spike, and as siliques were formed spread to these (Fig. 12). As the seed matured, the fungus grew from the lesions on the pods into the seeds. In this way it was possible actually to secure seeds from the vicinity of lesions, which, when plated out, gave rise to typical dry-rot colonies. In fact, so badly infected were these seeds (most of which were in addition immature) that in one instance 20 per cent. of infection was secured.

CULTURAL AND MORPHOLOGICAL CHARACTERS OF THE CAUSAL ORGANISM.

Dry-rot was determined by Kirk (5) as being due to the fungus *Phoma Napobrassicae* Rostr., but no cultural or comparative work was undertaken by this writer. As several *Phomas* have been recorded as attacking Brassicas, it was considered that substantiation of this diagnosis was necessary, for it is only when the specific nature of the organism is known that its economic importance in this and other countries can be accurately gauged. So a comprehensive programme of cultural work was undertaken. In addition, morphological and pathological comparisons were made with available "species" of *Phoma* occurring on Brassicas. These series of experiments embraced—

- I. (a) Inoculation into swede bulbs of all fungi found on swede and turnip seed, bulbs, and leaves, to determine parasitism.
- (b) Inoculation of cultures from abroad of related species isolated from Brassicas — namely, (i) *Phoma lingam*, (ii) *P. oleracea*, (iii) *P. Brassicae*, and (iv) *P. Napobrassicae*.
- II. Comparative cultures on various media of (i) *P. lingam*, (ii) *P. oleracea*, (iii) *P. Brassicae*, (iv) *P. Napobrassicae*. (foreign and New Zealand isolations).
- III. Comparisons of (i) microtome sections of pycnidia, (ii) spores taken from (a) cultures, (b) lesions on swede bulbs.

I. Inoculation Experiments.

Several hundred isolations of the dry-rot and other organisms have been made from lesions on leaves of swede, turnip, cabbage, kohlrabi, chou moellier, and rape, cotyledons of swede and turnip seedlings (obtained by germinating seed on germinator trays), bulbs of turnip and swede, stems of cabbage and broccoli, swede and turnip seed, and swede testas, embryos, flowering-spikes, and siliques. These have been cultured on various media, single spore or hyphal tip isolations made,



FIG. 12. DRY-ROT LESIONS ON SWEDE SILIQUAS. NATURAL SIZE.

Seed removed from beneath these lesions produced typical dry-rot colonies on culture media.

[Photo by H. Drake.]

and the inoculation experiments made from these resultant pure cultures. Usually the inoculations were conducted with portions of mycelium, owing to the fact that frequently cultures either remained sterile, or else produced pycnidia which did not freely sporulate.

Two methods were used: (i) swedes were grown in the glasshouse from sterilized seed sown in soil sterilized by live steam: (ii) swede bulbs from a crop known to be free from dry-rot were washed, the roots removed, surface sterilized, and placed in large sterile glass jars in the bottom of which was placed sterilized cotton-wool moistened with boiled water. The second method proved more satisfactory, as it was possible to keep the bulbs free from contamination, and to keep them in a fresh condition for three months. The following table shows the results of these inoculation experiments:—

TABLE II.—INOCULATIONS WITH DRY-ROT ORGANISM OBTAINED FROM VARIOUS PARTS OF SEVERAL BRASSICAS.

Source of Inoculum.				Result.	Source of Inoculum.				Result.
Leaves—					Bulbs—				
Swede..	..	..	+		Turnip..	..	..	+	
Turnip	..	..	+		Stems—				
Cabbage	..	..	+		Cabbage	..	..	+	
Kohl rabi	..	..	—		Broccoli	..	..	—	
Chou moellier	..	..	—		Swede (flowering)	..	..	+	
Rape ..	..	..	—		Seed—				
Wild turnip	..	..	—		Swede ..	..	..	+	
Seedlings (cotyledons)—					Turnip..	..	..	+	
Swede..	..	..	+		Rape ..	..	..	—	
Turnip	..	..	+		Testas (swede)	..	..	+	
Bulbs—					Embryos (swede)	..	..	—	
Swede..	..	..	+		Siliquas ..	..	..	+	

This shows that, with the exception of fungi isolated from lesions on leaves of kohl rabi, chou moellier, rape, wild turnip, broccoli-stems, rape-seed, and swede embryos, all were parasitic, producing as they did a readily noticeable rot within six days.

A similar series of inoculations was undertaken with cultures of *Phoma* from Brassicas obtained from abroad. These, listed under the names applied by those who forwarded the cultures, are given in the following table:—

TABLE III.—RESULTS OF INOCULATIONS WITH FOREIGN CULTURES.

Name of Organism.				Source of Culture.				Result.
<i>Phoma oleracea</i>	..	..		Centraal-Bureau, Holland	..	..	—	
<i>P. lingam</i>	..	..		"	"	..	+	
<i>P. Napobrassicae</i>	..	..		"	"	..	+	
<i>P. oleracea</i>	..	..		Dr. Quanjier, Holland	..	..	+	
<i>P. lingam</i>	..	..		Dr. Vaughan, Wisconsin, U.S.A.	..	..	+	
<i>P. Brassicae</i>	..	..		Pretoria, Union of South Africa	..	..	+	
From swede-bulb	..	..		Dr. Whitehead, Bangor, Wales	..	..	+	
From mangold-bulb	..	..		"	"	..	—	
From broccoli-stems	..	..		"	"	..	—	

These experiments were replicated many times, but the results did not vary.

II. Comparative Cultural Experiments.

The fact that similar lesions could be produced by apparently different species (as illustrated in Table III) necessitated a comparative study being made of these and the numerous isolations made from different substrata in New Zealand.

Several media were used for this purpose, as potato dextrose agar extract (200 grm. potato were minced and strained; to this extract were added 20 grm. powdered agar and 20 grm. dextrose dissolved in 500 c.c. water; the whole was then autoclaved at 10 lb. for twenty minutes), potato glucose agar extract (glucose replacing the dextrose in the previous formula), swede glucose agar extract (200 grm. of minced swede replacing the potato), swede agar extract (the glucose being left out), Brown's media (1), Brown's plus starch, and Brown's plus glucose, and crushed-oat agar (50 grm. crushed oats replacing the 200 grm. potato). Experience showed that, with the exception of Brown's media and its variations (on which all strains made poor growth and failed to fructify), all strains behaved similarly on the different media, but potato glucose proved most satisfactory in enhancing the differences in strains, consequently this last was used almost exclusively for comparative purposes.

Some 400 strains (in the sense of separate isolations) were used for these experiments.

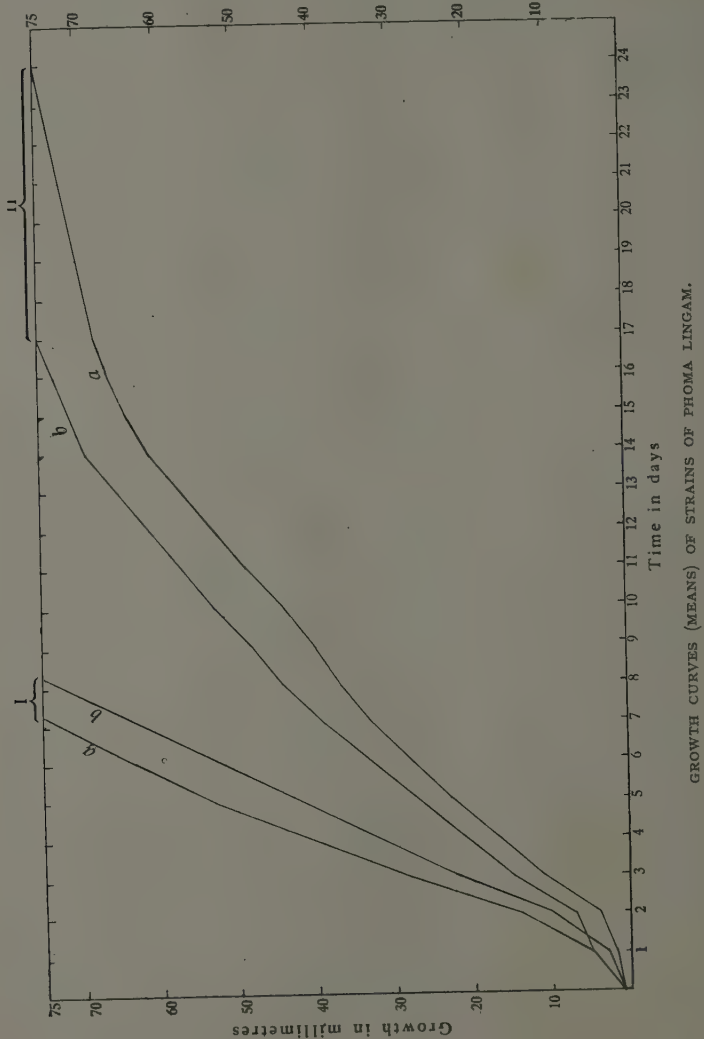
A preliminary experiment calculated to determine the effects of temperature and light was undertaken. In this a series of cultures from different cultural strains was prepared (on potato glucose agar extract) in quadruplicate. The plates were placed under the following conditions: (i) Room-temperature (18° – 21° C.); (ii) incubator at 21° C., saturated atmosphere*; (iii) incubator at 23° C.; (iv) incubator at 25° C.; (v) incubator at 30° C. In addition two out of the four plates of each strain were wrapped in four layers of heavy brown paper, in order to determine the effects (if any) of absence of light. Examinations were made daily, and results noted.

As a result it was found that there was no measurable difference between those cultures excluded from light and those exposed to normal lighting; nor was any marked difference in growth-rate apparent at any of the temperatures used, growth being approximately the same at 18° C. or 30° C. But marked staling becomes noticeable at 30° C., and (strangely enough) at 21° C. with those strains subject to staling; and at 21° C. the differences between strains tended to become more exaggerated. Therefore in the following comprehensive series this temperature was used.

All platings were made the same day, from stocks prepared ten days previously. Previous work had shown that numerous cultural strains existed among the numerous isolations made. Although apparent to the eye, it was to prove a difficult matter to define these differences so they could be detected by other workers. And after numerous methods had been tried out it was decided that the most

* Saturation was obtained by placing a wide dish filled with water immediately over the heating-coil in the bottom of the incubator. In this way it was possible to keep the media moist and prevent drying out, a trouble frequent in cultures kept over a period of weeks.

feasible one lay in plotting the growth curves of all isolations. The accompanying graph shows the means of such curves.



Examination of this graph shows that two related pairs of curves are present. These have been indicated by the symbols I and II, representing what may be termed groups of strains. Each

of these may in turn be divided into (a) and (b), separation being effected upon lesser differences. Rather than discuss at length the behaviour of these numerous strains the writer has tabulated them as under:—

DESCRIPTION OF STRAINS.

- I. Rapid-growing, reaching periphery of 75 mm. petrie dish in five to ten days. Growth in earlier stages densely olivaceous or fuscous.

IA. Reaches periphery in five to eight days. At first exhibits radial striae and concentric zones; later these become obliterated owing to density of growth. When viewed by transmitted light, mycelium is strongly fuscous, no olivaceous colour being present. Surface dingy grey or pallid-fuscous, often flocculent, sometimes zoned. Media (only on potato glucose agar extract) in five to seven days becomes stained pallid plum-colour. Margin of colony throughout remains even. Pycnidia numerous, chiefly peripheral, appearing in seven or more days. Non-staling. 10 per cent. of all isolations fall into this group. (Fig. 13.)

Sources of Strains.—Swede seedlings, seed, and bulbs; English and Danish seed.

Pathogenicity.—Weakly parasitic, taking twenty-two days to produce a lesion of a diameter greater than 5 mm. (Fig. 21.)

Notes.—One strain (473) saltates readily, giving rise to a sterile form otherwise identical; this in turn saltates, giving rise to the previous form. Some strains (notably 503 and 533) reach the periphery of the plate in five days: otherwise they are identical with the majority.

IB. Reaches periphery in seven to ten days. Strongly radially striate, and often concentrically zoned. Invariably light or dark olivaceous when viewed with transmitted light, the light-olive form sometimes approaching fuscous. Surface scanty tomentose or flocculent, seldom zoned. Media not stained. Margin of colony even throughout. Sterile, pycnidia being absent (save in rare exceptions, when, after long keeping, a few sparse peripheral pycnidia are produced). Non-staling. 8 per cent. of isolations fall into this group. (Fig. 14.)

Sources of Strains.—*Phoma Brassicae* from Pretoria, Union of South Africa; *Phoma lingam*, from Centraal-Bureau, Holland. Swede seedlings, testas, and bulbs; cabbage-leaves.

Pathogenicity.—Weakly parasitic, taking nineteen days to produce a lesion of a diameter of 5 mm. (Fig. 21.)

Notes.—Saltation frequent in the whole group, producing saltants which merge with group IA. In certain strains (notably 472, 473) saltation occurs regularly.

- II. Slow-growing, taking eighteen to twenty-four days to reach periphery of 75 mm. petrie dish. Of two types, separated chiefly by the nature of the margin, whether lobed or not. (See photographs.)

IIA. Lobed form. Slow-growing, taking eighteen to twenty-four days to reach periphery of plate. Marked staling occurs when colonies 30–40 mm. diameter (Fig. 15), then growth almost ceases for a few days; this is followed by the production of peculiar lobes from the periphery of the colony (Fig. 16). The lobes may be fan-shaped or dendriform: they are produced in numbers, and after their appearance make lateral growth, thus merging and adding considerable increase to diameter of the colony (Fig. 17). Staling again occurs following lobing, when growth is slowed to about 1 mm. per day, or may cease altogether (Fig. 18). A milk-like halo becomes apparent around staled colonies. Does not stain media (save one strain). Pycnidia (not numerous and usually non-sporulating) produced in five to ten days. 71 per cent. of isolations fall into this group.

Sources of Strains.—*Phoma lingam* from Wisconsin, U.S.A.; *P. oleracea* from Dr. Quanjer, Holland. Swede pods, seed, and bulbs, and turnip bulbs, from Southland, Canterbury, and Taranaki; cabbage-stems.

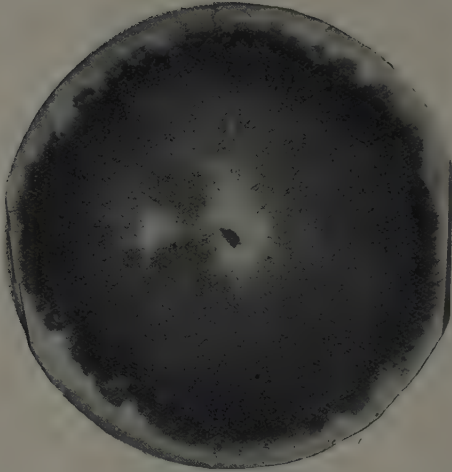


FIG. 13. CULTURAL CHARACTERS OF STRAINS OF PHOMA LINGAM. NATURAL SIZE. A FIVE-DAY CULTURE OF STRAIN IA.

[Photo by H. Drake.

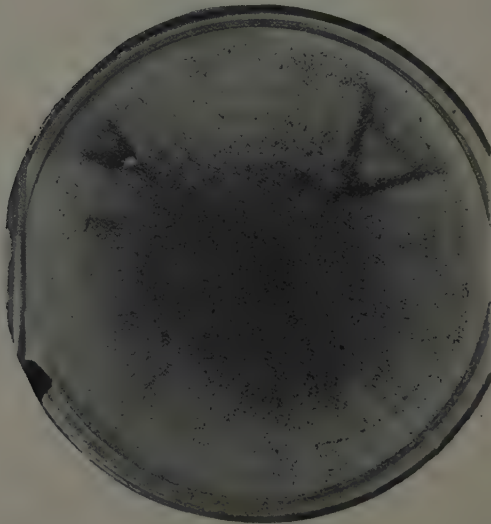


FIG. 14. NINE-DAY CULTURE OF STRAIN IB.

Note saltation.

[Photo by H. Drake.

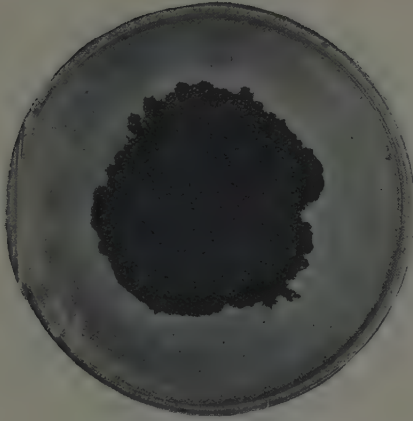


FIG. 15. CULTURAL CHARACTERS OF STRAINS OF PHOMA LINGAM. CULTURES OF STRAIN IIA.

Staled colony prior to lobing.

[Photo by H. Drake.

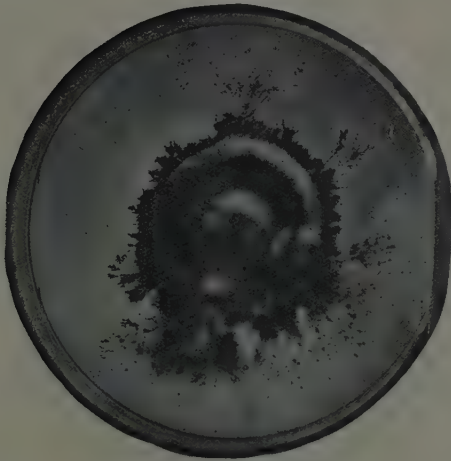


FIG. 16. CULTURAL CHARACTERS OF STRAINS OF PHOMA LINGAM. CULTURE OF STRAIN IIA.

Commencement of lobing.

[Photo by H. Drake,

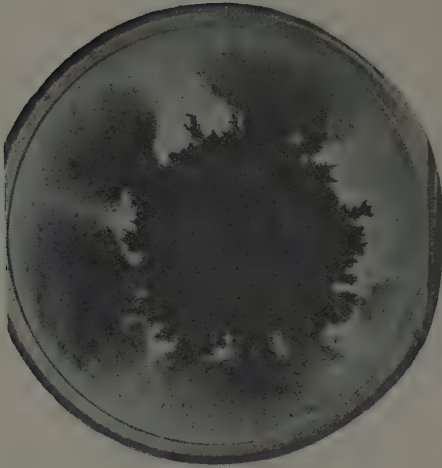


FIG. 17. CULTURAL CHARACTERS OF STRAINS OF PHOMA LINGAM. CULTURE OF STRAIN IIA.

Lateral fusion of lobes.

[Photo by H. Drake.

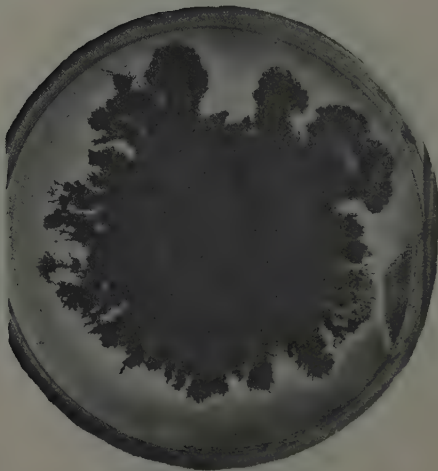


FIG. 18. CULTURAL CHARACTERS OF STRAINS OF PHOMA LINGAM. CULTURE OF STRAIN IIA.

Staled colony subsequent to lobing.

[Photo by H. Drake,

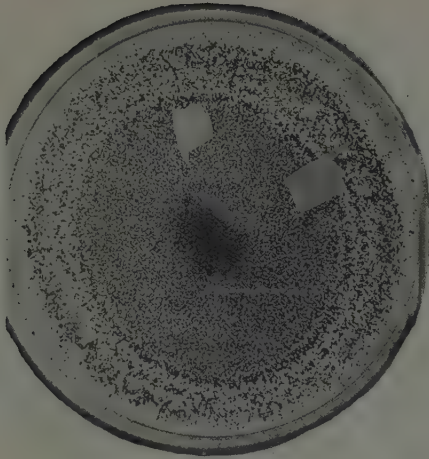


FIG. 19. CULTURAL CHARACTERS OF STRAINS OF PHOMA LINGAM. CULTURE OF STRAIN III.

Note intense pycnidial production.

[Photo by H. Drake.

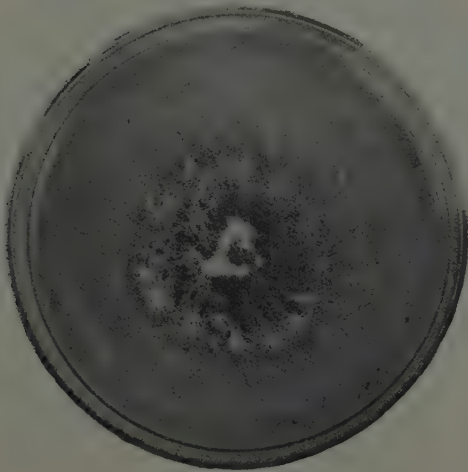


FIG. 20. CULTURE OF STRAIN III. STERILE SALTANT FROM THE PREVIOUS PYCNIDIATE FORM.

[Photo by H. Drake.



FIG. 21. LESIONS PRODUCED BY STRAINS 1A AND B. $\times \frac{1}{2}$.
Photo taken nineteen days after inoculation. Note
small size of lesions.
[Photo by H. Drake.]

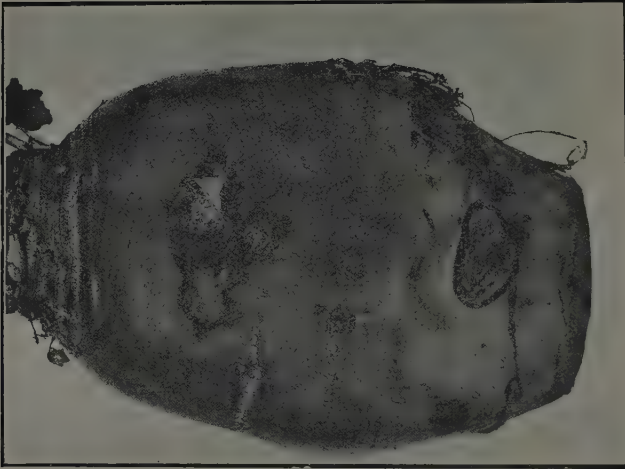


FIG. 22. LESIONS PRODUCED BY STRAIN 11A. $\times \frac{1}{2}$.
[Photo by H. Drake.]

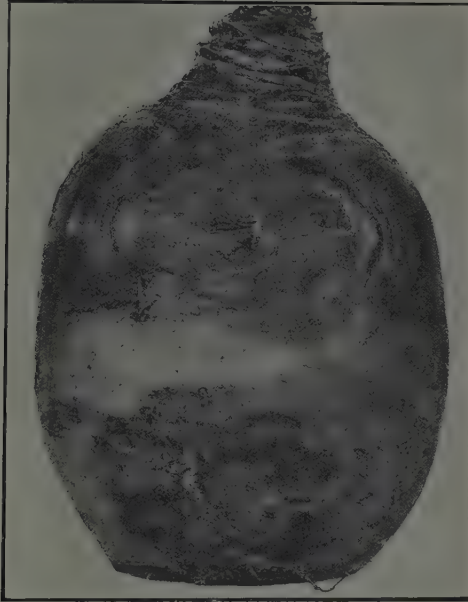


FIG. 23. LESIONS PRODUCED BY STRAIN IIA. $\times \frac{1}{2}$.

[Photo by H. Drake.]



FIG. 24. LESIONS PRODUCED BY STRAIN IIA. $\times \frac{1}{2}$.

Note that lesions in Figs. 22, 23, and 24 differ considerably from one another, yet all were taken 19 days after inoculation with the same strain.

[Photo by H. Drake.



FIG. 25. LESION PRODUCED BY STRAIN IIB. $\times \frac{1}{2}$.

Photo taken 19 days after inoculation.

[Photo by H. Drake.

Pathogenicity.—Strongly parasitic, all isolations tried producing characteristic dry-rot lesions in five days. Spores (obtained from one or two strains which sporulated readily) also produced characteristic lesions, but required twelve days before becoming apparent. Pycnidia found in bulbs after twenty-two days. (Figs. 22, 23, 24.)

Notes.—A strain which is slow-growing, and soon stales. The lobing is characteristic, but not always apparent. This is the strain chiefly responsible for dry-rot in New Zealand.

- IIb. Non-lobed form. Similar to IIA, save that lobing is not apparent, and the growth somewhat more rapid. Staling becomes marked only towards the end of the growth-curve. The group is somewhat heterogenous, as some strains are pycnidiate, others sterile: some olivaceous, others partly so; others again pallid or almost colourless. Some stain media, others do not. 6 per cent. of isolations fall into this group.

Sources of Strains.—*Phoma Napobrassicae* from Centraal-Bureau, Holland; strain from swede bulb, from Bangor, Wales; mummied bulbs from Gore, Southland; bulbs of swedes from Southland and Canterbury; cabbage-leaves.

Pathogenicity.—Similar to IIA. (Fig. 25.)

Notes.—A heterogenous group, separated chiefly by the non-lobing character. Saltation not infrequent, giving rise to Ib and IIA.

- III. Growth-curve similar to Ib. Scanty white mycelium, scarcely discernible on the media; dense pycnidial formation in five days, abundant pink spore-masses produced in six days. Pycnidia may be in zones or not. Non-staining. 0.25 per cent. of strains fall into this group. (Fig. 19.)

Sources of Strains.—*Phoma oleracea* from Centraal-Bureau, Holland. Swede seedlings and leaves.

Pathogenicity.—Constantly negative, despite numerous inoculations.

Notes.—A distinct saprophyte strain, readily recognized by its copious early sporulation. Freely saltates, giving rise to a sterile mycelial form (Fig. 20) which resembles one strain in IIb. This in turn saltates, giving rise again to sporulating type. It is inserted here as of interest because it was sent as a culture of *P. oleracea* from Centraal-Bureau.

Numerous other strains differing considerably from those described have been isolated from seed and bulbs of swedes and turnips, but, as all have proved incapable of infecting swede-bulbs, they will not be considered further.

From this it will be seen that the organism causing dry-rot in New Zealand consists of a number of distinct groups of strains, which, irrespective of the media on which they have been grown, remain constant save where saltation tends to link up certain strains of different groups. Further, it will be noted that the foreign cultures received from abroad fall into four of these groups, as under:—

Group.	Source of Culture.
IIA ..	<i>Phoma lingam</i> from Wisconsin.
IIA ..	<i>P. oleracea</i> from Dr. Quanjer, Holland.
IB ..	<i>P. lingam</i> from Centraal-Bureau, Holland.
IB ..	<i>P. Brassicae</i> from South Africa.
IIb ..	<i>P. Napobrassicae</i> from Centraal-Bureau, Holland.
IIb ..	Strain from swede from Bangor, Wales.
III ..	<i>P. oleracea</i> from Centraal-Bureau, Holland.

This shows that cultures of the four "species" *Phoma lingam*, *P. oleracea*, *P. Napobrassicae*, and *P. Brassicae* agree with strain groups similar to those isolated from swede, cabbage, and turnip bulbs, seed, and leaves in New Zealand.

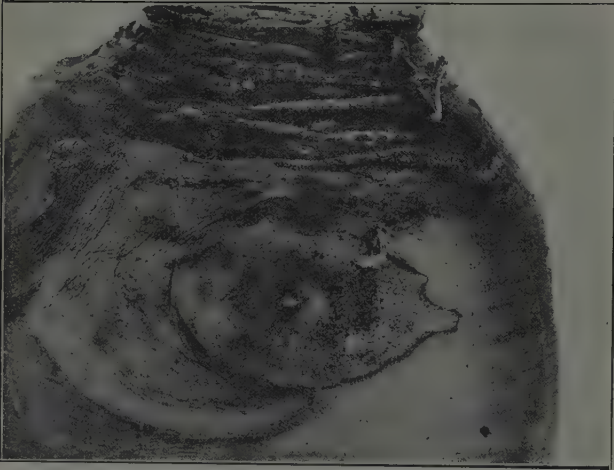


FIG. 26. LESION PRODUCED AS A RESULT OF INOCULATION WITH SPORES OF STRAIN IIA. $\times \frac{1}{2}$.

Note pycnidia.

[Photo by H. Drake]

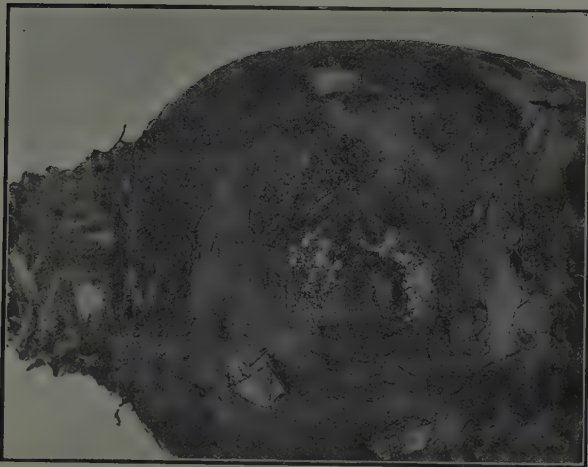


FIG. 27. FRUCTIFICATIONS OF A FUSARIUM WHICH COMMONLY FOLLOWS DRY-ROT. $\times \frac{1}{2}$.

Note manner in which characteristics of lesions have been destroyed.

[Photo by H. Drake.]

Further, a strain labelled *Phoma lingam* from Wisconsin (isolated from cabbage by Dr. Vaughan) does not agree with one labelled *P. lingam* (isolated from cabbage at Wisconsin by Dr. Walker) from Centraal-Bureau, Holland; nor does the one labelled *P. oleracea* from Dr. Quanjer, Holland (isolated from cabbage) agree with the one labelled *P. oleracea* from Centraal-Bureau (isolated from cabbage by Dr. Sluiter, Holland).

From this evidence it will be seen that little reliance can be placed upon cultural comparisons as a means of specific delimitation. Therefore, as accurate diagnosis was not possible, despite an intensive cultural comparison of the organism causing dry-rot with foreign cultures, an intensive morphological study of the causal organism was undertaken.

III.] Morphological Comparisons.

In addition to abundant pycnidial material secured for comparison from bulbs and leaves of turnips and swedes, as well as those obtained from different strains on media, material was obtained from abroad: as *P. Napobrassicae* on swede-bulb from Dr. G. H. Pethybridge, England; *P. lingam* on leaves, stems, and siliquas of cabbage from Dr. Vaughan, Wisconsin, U.S.A.; *P. lingam* from decayed bulb of swede collected in Finland by P. A. Karsten in 1891 and forwarded by Dr. Shear, Department of Agriculture, Mycological Exchange, U.S.A.; *P. Brassicae* on cabbage from Department of Agriculture, Pretoria, Union of South Africa.

Material was embedded in paraffin and cut 5 and 10 microns in thickness on the microtome. Spores were also mounted direct on slides (previously smeared with a fixative), fixed and stained in iron alum hæmatoxylin. In this way large numbers were secured and accurate measurements made possible.

It was soon realized that comparison of this material was complicated in that two distinct types of pycnidia occur: (i) one with a comparatively thin, light-coloured wall, small size, depressed shape, submerged habit, abundant in living leaves, siliquas, stems, bulbs, and cultures (Fig. 28); (ii) one with a thick, dense, dark-coloured wall, larger in size and superficial, present on dead tissues of cabbage-stems, old mummied swede-bulbs and the like (Fig. 29). Thus it appeared as if two distinct organisms were present, but cultures (from swede-bulbs) of type (ii) gave rise to colonies on media identical with colonies arising from type (i), and which later produced on the media pycnidia of type (i). Furthermore, on inoculation both proved equally parasitic. This diversity in form has already been pointed out by Henderson (4).

Comparisons of sections of the material discussed above as well as pycnidia produced in culture by the five strain-groups discussed in a previous section, showed that considerable variation (exclusive of the two types discussed in the previous section) occurred not only in size, shape, thickness of wall, and position of the pycnidia, but in length and diameter of the spores. However, as these are not constant for any one strain, but occur to an equal extent in different cultures of the same strain, they cannot be considered as being of any systematic value; thus morphologically all must be

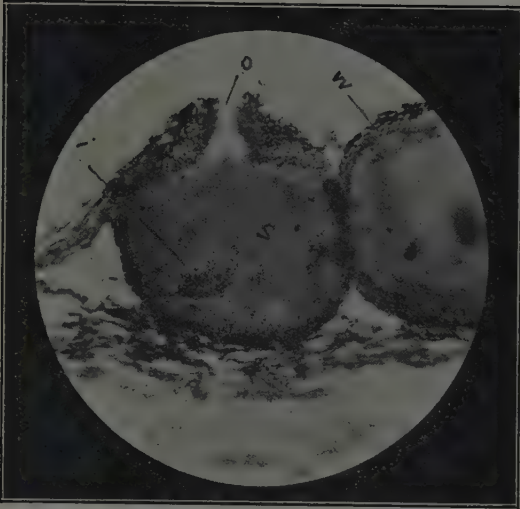


FIG. 28. SECTION OF PYCNIDIUM (TYPE I) OF PHOMA LINGAM IN SWEDE-BULB. $\times 125$.

Note "islands" (s) of pseudo-parenchyma, thin wall (w), spore mass embedded in mucilage (s), and papillate ostium (o).

[Photomicrograph by Writer.

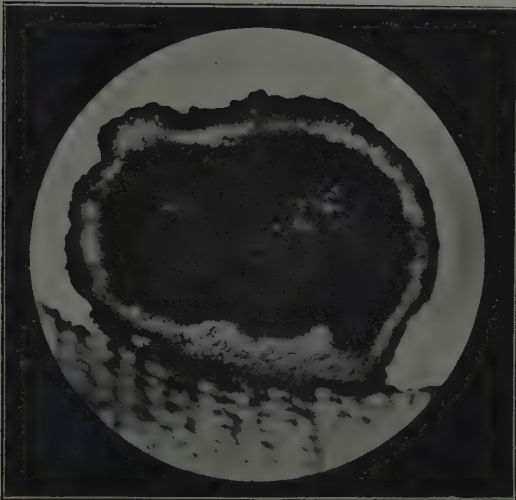


FIG. 29. SECTION OF PYCNIDIUM (TYPE II) OF PHOMA LINGAM ON OLD MUMMIED SWEDE-BULB. $\times 125$.

Note thick wall and fact that the pycnidium is completely naked. Section not median so ostium not shown.

Photomicrograph by Writer.

classed as belonging to the same species. It was noted on numerous bulbs that certain spore-masses produced were pink, others again being white. But cultures made from both produced colonies alike, and indistinguishable one from the other. And these resultant cultures produced pycnidia which in turn produced white spore-masses. So the colour of the spore-mass cannot be considered as being of any value. This, too, has been commented on by Henderson (4, p. 386) who states that from pycnidial type (i) the spores "are discharged through a distinct ostiole at the upper surface in a snake-like stream, which has been found to be either pinkish or whitish in colour."

As comparison of the pycnidia of several strain-groups has shown them to be morphologically identical, even when labelled *Phoma lingam*, *P. Napobrassicae*, *P. oleracea*, and *P. Brassicae*, it is evident that these are synonyms of a single species. Examination of the original descriptions shows, moreover, that they equally well may be applied to any or all of these. Consequently the writer considers all are synonyms of one species.

The prior name applied to the organism causing dry-rot is *Phoma lingam* (Tode) Desm., consequently this must be the name for this pathogen.

Phoma siliquastrum Desm. was applied by Desmaziere to a form found on the siliques and peduncles of Brassicae. The same form has been recorded (12, p. 31) as attacking stems of flowering swedes in England in 1923. As has been shown, the organism on stems and siliques of swedes in New Zealand is a strain of the dry-rot organism. Further, the original description agrees with that of *P. lingam*, therefore this name must be considered as a synonym of this species, as has been pointed out by Henderson (4).

Phoma oleracea Sacc. was in 1880 applied to a form on cabbage, and related varieties of *Brassica oleracea*. Culturally and morphologically this is identical with strains of *P. lingam*, and must therefore be considered a synonym of this species.

Phoma Brassicae Sacc. was also named from decayed cabbage, and has by previous workers been shown to be a synonym of *P. oleracea* (*P. lingam*).

Phoma Napobrassicae Rostr. was in 1892 described from swede-bulbs by Rostrup. And as has been shown, this is the name by which the organism causing dry-rot has been known in New Zealand since 1905, and by which it has been known in Europe and Britain since Rostrup's publication in 1892. A culture from Centraal-Bureau (isolated by Miss Weber, Denmark) sent as *P. Napobrassicae* falls into strain-group IIB. Pycnidia from these cultures, and from swede-bulbs sent by Dr. Pethybridge, England, are morphologically identical with others labelled *P. oleracea* and *P. lingam*. Therefore this must be considered as a synonym of *P. lingam*.

Phyllosticta Brassicae (Curr.) West., recorded on cabbage-leaves, is, judging from the description, merely the form of *P. lingam* on leaves.

Type (ii), with a thick-walled, laminated pycnidium, occurring on old stumps of cabbage, mummied bulbs of swede and the like, has been named *Plenodomus lingam* (Tode) v. Hohn. (13). This combination is recognized by Diedicke (3). Nevertheless, as has been shown, this is but a thick-walled form of *Phoma lingam*. (Fig. 29.)

The following amended diagnosis has been drawn up from the material discussed above.

Phoma lingam (Tode) Desmaziere, *Ann. Sci. Nat.*, ser. iii, vol. 2, p. 280, 1849.

P. siliquastrum Desm., *l.c.*

Phyllosticta Brassicae (Curr.) West., *Bull. Acad. Brux.*, p. 397, 1851.

P. oleracea Sacc., *Mich. Comm. Myc.*, vol. 2, p. 91, 1880.

Aposphaeria Brassicae Theum, *Hedw.*, vol. 12, p. 189, 1880.

Phoma Brassicae Sacc., non Thuem, *Syll. Fung.*, vol. 3, p. 119, 1884.

P. Napobrassicae Rostr., *Tidsskrift. for Landokonomi*, vol. 11, p. 330, 1892.

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Dry-rot, black-leg, foot-rot, turnip-phoma rot, phoma-rot, turnip-rot, fallsucht, krebstrunke.

Pycnidia variable in size and shape, globose, depressed-globose, lenticular or flask-shaped, 130–340 microns in diameter; ostiole 15–20 microns in diameter, papillate or not; wall coloured brown or (when naked) black, 12–18 microns in thickness, save at the apex where frequently considerably thickened. In type (ii) the wall is laminated, black, and thickened up to 50 microns. Conidiophores hyaline, cylindrical, continuous, inevident, up to 8 microns in length. Spores hyaline, continuous, elliptical, sometimes slightly curved, ends bluntly rounded, less commonly acuminate, $3.5-6 \times 0.8-2$ microns; with a large central nucleus.

Hosts.—On leaves, flowering-stems, siliques, and bulbs of swede (*Brassica rutabaga*); leaves and bulbs of turnip (*Brassica Rapa*); stems and leaves of cabbage (*Brassica oleracea*), in New Zealand. On cabbage, cauliflower, Brussels sprouts, kohlrabi, kale, rape, swedes, and turnips in Europe and North America, and cabbage in Australia and South Africa. In addition Henderson (4) in North America has successfully inoculated with *P. lingam* white mustard, black mustard, charlock, wild and cultivated radish, tumble-mustard, hedge-stock, sweet alysium, and shepherd's-purse. It must be stressed that it has not been recorded as being found on these hosts in nature, however, nor in New Zealand on other than the hosts recorded, though fungi on numerous others were tried out.

Massee (11, p. 407) records *Phoma Napobrassicae* as attacking mangolds, but this is an error, the heart-rot of this host being caused by *Phoma Betae*.

Distribution.—Europe, Britain, North America, South Africa, Australia, New Zealand.

SYMPTOMATOLOGY.

The appearance of the disease varies considerably, according to the part of the host attacked, and even on the same part (as bulb) variation is considerable. It was thought that it might be possible to determine the presence of any particular strain by the type of lesion it produced. So when isolations were made from different types of bulb-lesions, the

bulbs from which the isolations were made were kept, and on each lesion was pinned a label bearing a number corresponding to the number of the culture. Examination of cultures when it was possible to determine the strain by cultural characters showed that determination of strains in this manner was not possible, as the same strain may, even on the same variety of swede, produce a considerable diversity of lesions. Inoculations of different strains into swede-bulbs gave in general the same conflicting results, save those produced by group I, where parasitism was weak, and the lesions consequently small.

It was also noted that a large number of lesions bore abundant pycnidia, while others, apparently identical otherwise, were sterile. But isolations from these showed that the presence or absence of pycnidia could not be considered as possessing any value, as from both types similar strains were isolated, and when both were inoculated into swede-bulbs they formed pycnidia freely or remained sterile. Finally, it was found that a small percentage (2.0 per cent.) of lesions resembling closely those produced by *Phoma lingam* (save that they were more deeply depressed and softer to the touch) were caused by a *Fusarium*, which on inoculation proved definitely parasitic. It was found that this organism frequently followed (in the field) *P. lingam* infection, and was a factor largely responsible for the rapid rot of such bulbs. The *Fusarium* has not yet been identified.

Appearance on Leaves.—On leaves of swedes and turnips small circular spots, slightly depressed, 1–10 mm. diameter, are commonly produced. These at first are tobacco-brown, but often become ashy-grey in the centres. The margins are frequently slightly elevated, and often tinged with purple. Pycnidia (type i) are abundant, amphigenous, though chiefly epiphyllous, black, submerged, and frequently arranged in concentric zones. On cabbage-leaves the spots often merge, forming cuneiform areas, ashy-grey in colour, extending from the apex to midrib of the leaf.

Stems.—On cabbage-stems a brown or grey discoloration is produced, followed usually by death of the plant. On the exterior of the killed tissues abundant superficial black pycnidia (type ii) are produced. On swede flowering-spikes, ashy-grey, elliptical depressed lesions, up to 100 mm. or more, are formed, and in these are present immersed black pycnidia (type i).

Siliques.—Ashy-grey depressed spots, with black or purple margins, are produced in abundance; pycnidia similar in appearance to those on leaves are abundant. Internally a flocculent mycelium is often present, extending to and surrounding seeds in the vicinity of lesions.

Seeds.—The pathogen is not visible externally on swede or turnip seed, even when these are taken from the vicinity of siliqua lesions.

Bulbs.—Early infection of the bulb occurs in the region of the neck. Here small green lesions are produced, which contrast with the usual purple colour of this portion of the bulb. The affected area soon becomes slightly sunken, and as the lesion increases in diameter the centre becomes fuscous and finally ashy-grey or bay-brown. Dark-brown or almost black lesions are not uncommon, especially on the lower white portion of the bulb, where pigmentation of the epidermis is wanting. In bulbs less than half the normal diameter spread of the disease is rapid, so that the whole region beneath the crown becomes discoloured. Sections show the flesh to be discoloured and altered to

a "punky" consistency, consequently the area of penetration is conspicuous and readily delineated.

At a later stage the lesions become more numerous, then being scattered over the whole of the exposed portions of the bulb. At first small, they rapidly enlarge, spreading mostly radially, and often merge. Penetration is not so marked, the diseased area often being confined to a layer extending 15 mm. or less into the flesh.

In bulbs nearing maturity the lesions may be very numerous, but are at this stage small, slightly or not depressed, and confined usually to the crown.

Pycnidia (No. i type) are often abundant in lesions, appearing fourteen to twenty-five days after infection. They are submerged, black, and often concentrically arranged. On old bulbs they become exposed, and superficial through the falling-away of the overlying epidermal tissues. Other lesions of the same type frequently remain sterile, but, as has been shown, this is of no significance from a diagnostic viewpoint.

When inoculations are made in bulbs kept under sterile conditions, lesions produced by group IIA and B rapidly spread until in twenty-one days they may have a diameter of 75-100 mm. The colour of such is usually some shade of brown, and pycnidial formation scanty; when present they appear only after twenty days, or even much later. Group IA and B produces small lesions which do not enlarge to any extent, even after long keeping of the inoculated bulbs.

VARIETAL RESISTANCE.

As the following particulars show, no swede variety at present grown in New Zealand can be considered to be dry-rot-resistant.

In 1918 Levy (7) carried out trials with twenty-nine swede varieties. He found on examination late in the season that the percentage of infected bulbs ranged from 73 to 92 per cent. A further series was carried out on Weraroa Experimental Farm in 1922-23, fifty-three varieties being experimented with. Although the season was a dry one and infection consequently slight, inspection showed dry-rot to be present to a slight extent in all. Since this period field observations have been made each season, and all varieties noted to be infected to a greater or less extent.

Under certain conditions certain varieties may appear to be resistant, but in the light of our present knowledge this apparent resistance may be attributed to other factors, as dry-weather conditions, or the absence of the dry-rot organism in the seed-line used. For, as is shown in Table I, out of the commercial lines tested, five were found to be disease-free.

DEVELOPMENT OF THE PYCNIDIA.

As numerous microtome preparations have been made both from cultural material and material taken from bulb-lesions, excellent opportunity was afforded for studying the development of pycnidia and spores, and the mechanism (as it were) of spore-discharge.

A copious mycelium is produced following infection, and the freely septate hyphæ penetrate into and between the host cells. Here

chlamydospores are produced in abundance, by the enlargement of certain groups of contiguous cells, and thickening of their walls. Chlamydospores serve as structures adapted to carry over a fungus through some unfavourable period, but other than serving to help the organism to persist in mummied bulbs for a period they do not appear materially to prolong the carry-over period. Shortly "knots" of hyphæ become aggregated immediately beneath the epidermis, and frequently form quite a definite stromatic structure in this region, producing the frequent dark colour observed in lesions. These at first are of single hyphæ, but they soon become more densely compacted, and finally become quite pseudoparenchymatous. The outer layer especially becomes distinctly darker in colour, the cells larger and light brown in colour. Then one or several areas appear in which gelatinization of the central tissues takes place, but splitting or rupture does not occur. In these centres conidiophores made their appearance and quickly produce abundant spores. As yet no ostium is present. Further and more general gelatinization then takes place until the intervening tissues between the original centres disappear (save peripherally, where certain "islands" persist for some time). The mucilaginous matter thus produced swells considerably, distending the outer walls, and the pycnidium begins to take a definite shape.

Pressure being exerted, expansion occurs in the direction of least resistance—that is, towards the periphery of the bulb—consequently the pycnidium tends more and more to become flask-shaped from its original globular appearance, and an elongated neck-like portion of tissue, contiguous with the wall, extends towards the exterior. Here, owing to the gradual increase in pressure through enlargement of the pycnidia, small dome-shaped pustules become noticeable. These rupture, and the pale-brown elongated apices of the pycnidia become visible. These on exposure become darker, and, finally, quite or almost black.

The ostium then becomes formed by gelatinization of the central portion of the neck-like projection, and becomes internally lined with periphyses. (Fig. 28, *o*.) As the epidermis falls away from the exterior of the pycnidium, increase in thickness occurs, accompanied by thickening and darkening of the walls, until finally, in old and desiccated bulbs, where the pycnidia have become completely exposed, the latter are seen to possess thick, almost black walls, then agreeing with type (ii) described above and forming the character of *v. Hoehnel's* genus *Plenodomus*. (Fig. 29.)

Small, isolated, usually peripheral "islands" of undifferentiated tissue (Fig. 28, *i*) have been mentioned as remaining ungelatinized when general gelatinization of the central pseudoparenchyma occurred. This tissue as the pycnidium ages gradually becomes gelatinized in turn, thus serving to keep up a constant supply over a long period of the gelatinous matrix so essential to spore-dissemination.

The spores are produced from conidiophores—short, cylindrical, hyphal-like projections—lining the inner wall of the pycnidium. At maturity they are chiefly elliptical, colourless, and uninucleate.

The spores germinate readily in potato or swede glucose extract, provided they are taken from fresh exudate. As has been shown, they are capable of remaining viable for only a short period, if the gelatinous matrix in which they are embedded is removed.

LABORATORY EXPERIMENTS IN CONTROL.

As dry-rot is seed-borne it naturally follows that seed-treatment would be the efficient method of combating it.

Consequently the available literature dealing with this and related diseases was searched to ascertain whether a suitable control had been evolved. Henderson (4, p. 420), dealing with *P. lingam* on cabbage, claimed as a result of experiments that a satisfactory treatment consisted in immersing the seed for twenty to twenty-five minutes in 1-200 solution of formalin, followed by washing in clean water, although complete control was not claimed. Walker (14) claimed that immersion in 1-240 formalin or 1-1,000 HgCl_2 for thirty minutes, followed by rinsing, was a partial controllant, but often unsafe, owing to the detrimental effect upon the germination of the seed. The same author (15) claimed that complete control could be obtained by thirty minutes immersion in water heated to 50° C., with but slight injury to the seed provided this was less than six months old.

Thus we find that in North America both chemical steeps and hot-water treatment have been recommended for combating a cabbage-disease caused by the dry-rot organism. Complete control is claimed only with the latter, but this is advocated only when the seed is known to be fresh, on account of the danger of lowering germination.

Experiments conducted in this laboratory to ascertain whether these American treatments could be utilized are given below, in addition to numerous others. In this work the treatments were carried out by Mr. J. C. Neill, Field Mycologist, the culture-work being performed by the writer. After treatment the seed was plated out in the manner described for determining the presence of dry-rot in a seed-line, and examination made each day until ten days, the number of *P. lingam* colonies present in 1,000 seeds serving as an indicator of the efficacy of the method.

Chemical Treatments.

Treatment I: Mercuric Chloride.—One thousand seeds known to contain approximately 5 per cent. of the disease were soaked in mercuric chloride 1-1000, and mercuric chloride 1-1000 to which had been added 1 per cent. of concentrated commercial hydrochloric acid, for ten, twenty, thirty, forty, fifty, and sixty minutes respectively. The following table shows the results obtained:—

TABLE IV.—EFFECTS OF MERCURIC-CHLORIDE TREATMENTS.

Time of Immersion, in Minutes.				Solution.		Percentage of Germination of Seed.	
				HgCl_2 .	$\text{HgCl}_2 + \text{HCl}$.	HgCl_2 .	$\text{HgCl}_2 + \text{HCl}$.
				Colonies.	Colonies.		
Control	..	..	..	47	48	82	82
10	..	..	..	42	40	80	78
20	..	..	..	38	38	78	79
30	..	..	..	27	22	74	71
40	..	..	..	28	22	63	55
50	..	..	..	25	15	51	30
60	..	..	..	19	7	42	17

Table IV shows that even with sixty minutes soak in 1-1000 mercuric-chloride solution the disease was not eliminated, and the effect of this was to reduce seed-germination from 82 per cent. to 42 per cent. With the acidulated solution the percentage of the disease was reduced, but the effects upon germination were more serious, as at 60 minutes germination was reduced in one series to 17 per cent. As results were so unsatisfactory—for the presence of seven infected seeds in 1,000, which was the lowest number of diseased seeds obtained, cannot be considered as being satisfactory, for 0.1 per cent. or less in any crop in a dry-rot season is sufficient to eliminate that crop—the formaldehyde treatment, which American workers admitted did not control the disease completely, was ignored.

Treatment II: Mercurio-phenol Compounds.—A series of experiments was undertaken with Uspulun, Germisan, and Semesan. In all cases the standard strength (0.25 per cent.) was used. One thousand seeds from the same sample as used in the previous experiment were soaked in these solutions for one hour and plated.

TABLE V.—EFFECTS OF MERCURIO-PHENOL COMPOUNDS.

Solution.				Colonies.	Percentage of Germination of Seed.
Control	..	..	..	42	82
Uspulun	..	..	..	47	80
Germisan	..	..	..	43	78
Semesan	..	..	..	47	81

As these experiments showed that the substances used had no appreciable effect on the fungus—unless a stimulating one—they were discarded without further experiment. A similar series, in which the soaking was extended to two hours, gave almost precisely the same results.

Hot-water Treatments.

Therefore a series of hot-water treatments was undertaken. The previous season, before we had evolved a satisfactory method of determining the presence of the disease in the seed, Mr. Neill treated a number of samples both of farmers' seed and a quantity obtained from a merchant. He treated this seed in separate lots, keeping careful records of the germination, using as a treatment a method he had previously found so successful in the control of a seed-borne disease of wheat, *Ustilago Triticæ*. This method consisted in presoaking the seed for from six to eight hours at 54° F., then dipping in hot water held at 130° F. for ten minutes. The seed was then spread out and dried. Careful record was kept of the behaviour of each of these lots on the areas where they were sown, but the results obtained were so conflicting as to render these worthless from a control experimental point of view; but one factor was clearly brought out—namely, that treated seed at first was slower in germination than untreated, coming through the ground over a much longer period; eventually, however, such crops caught up and passed (in appearance, at least) untreated lines sown side by side; and in one case (in Taranaki) the crop of

3 acres from treated seed was entered for the district swede competitions. The crop weighed out at 69 tons 18 cwt., thus easily winning the competition.

When a method of detecting the presence of the fungus in the seed was accurately worked out, experiments were made with the line used in the previous experiments, containing approximately 5 per cent. of the disease. One thousand seeds were treated as follows: Presoaked eight hours at 52° F., dipped ten minutes, 130° F.: result, thirty-four colonies. This showed the method was worthless as a controllant of the disease.

Next a ten-minutes dip at different temperatures without presoak was tried, 1,000 seeds for each series being used. The results are interesting, especially when seed-germination figures are taken into consideration with the killing temperature of the pathogen.

TABLE VI.—HOT-WATER TREATMENT: TEN MINUTES DIP WITHOUT PRESOAK.

Treatment.	Colonies.	Percentage of Germination of Seed.	Treatment.	Colonies.	Percentage of Germination of Seed.
Control ..	48	82	133° F. ..	3	33
125° F. ..	14	59	135° F. ..	1	14
127° F. ..	11	55	137° F. ..	0	12
130° F. ..	9	48	139° F. ..	0	3
131° F. ..	10	46	141° F. ..	0	0

Walker's treatment (15) was then tried. This consisted in immersing the seed for thirty minutes in hot water held at 121° F. In ten days twelve colonies were obtained.

A fungicide plus hot water was then decided upon. A 0.25-per-cent. solution of Semesan was maintained at different temperatures, as under. Seed soaked for one hour (1,000 in each series) and plated.

TABLE VII.—HOT SEMESAN SOLUTION: SEED IMMERSSED ONE HOUR.

Treatment.	Colonies.	Treatment.	Colonies.
Control ..	48	105° F. ..	17
95° F. ..	49	110° F. ..	1
100° F. ..	37	115° F. ..	0

This solution gave such satisfactory results at the higher temperatures that it was decided to work out a comprehensive series to ascertain the maximum killing-point of the pathogen with the minimum degree of injury to the seed.

Two series were run, one at 110° F., the other at 115° F.; and two different lines of seed were used, one (A) being of moderate germination but containing approximately 5 per cent. of diseased seed, the other (B) being of high germination but containing 1.5 per cent. of diseased seed. Two thousand seeds were used in each series, instead of the customary thousand, the germination figures given being the mean derived from twenty lots. Each series was dipped for ten, twenty, thirty, forty, fifty, and sixty minutes respectively

TABLE VIII.—HOT SEMESAN HELD AT 110° F. FOR DIFFERENT TIMES.

"A" Seed.			"B" Seed.		
Treatment.	Colonies per 1,000 Seeds.	Percentage of Germination of Seed.	Treatment.	Colonies per 1,000 Seeds.	Percentage of Germination of Seed.
Control ..	47	85	Control ..	15	92
10 minutes ..	24	80	10 minutes ..	2	92
20 " ..	20	77	20 " ..	1	92
30 " ..	4	78	30 " ..	0	92
40 " ..	4	76	40 " ..	0	92
50 " ..	1	78	50 " ..	0	92
60 " ..	2	77	60 " ..	0	92
Water only at 110° F. for 60 minutes	23	85	Water only at 110° F. for 60 minutes	10	92

TABLE IX.—HOT SEMESAN HELD AT 115° F. FOR DIFFERENT TIMES.

"A" Seed.			"B" Seed.		
Treatment.	Colonies per 1,000 Seeds.	Percentage of Germination of Seed.	Treatment.	Colonies per 1,000 Seeds.	Percentage of Germination of Seed.
Control ..	48	85	Control ..	15	92
10 minutes ..	10	78	10 minutes ..	0	85
20 " ..	7	60	20 " ..	0	85
30 " ..	1	58	30 " ..	0	86
40 " ..	1	57	40 " ..	0	82
50 " ..	0	50	50 " ..	0	80
60 " ..	0	46	60 " ..	0	80

These results show that complete control was not obtained with "A" seed at 110° F. even after a sixty-minutes soak; whereas with the same seed soaked at 115° F. for fifty minutes complete killing of the pathogen was secured. With "B" seed, control was obtained at 110° F. with a thirty-minutes soak, and in ten minutes at 115° F.

With "A" seed the germination percentage was reduced from 85 to 77 with the sixty-minutes soak at 110° F.; from 85 to 46 at 115° F. The "B" seed, on the other hand, suffered no appreciable depreciation in germination at 110° F. for sixty minutes, but was depressed by 10 per cent. at 115° F. for sixty minutes.

FIELD EXPERIMENTS IN CONTROL.

To test the efficacy of this treatment in the field a number of 14 oz. bags each of "B" seed treated at 115° F. for sixty minutes, and untreated seed from the same line, were forwarded to approximately one hundred farmers and overseers of departmental experimental farms throughout New Zealand. Details as to exact procedure to be adopted was issued with each lot.

A recent survey of the sown areas by Mr. Neill showed that, with certain exceptions, the results obtained could not be considered as possessing any experimental value. For many farmers neglected to clean their drills before sowing the treated seed; others did not remember which were the treated and which the untreated areas. In most

cases, however, the experiment was negatived in that, during cultural operations subsequent to sowing, cultivators were driven direct from untreated to treated areas, without attempts being made to minimize the danger of contamination. Other factors operating against success were wandering stock, rodents, and men walking between treated and untreated areas. Such contaminations are inevitable where treated and untreated areas are grown together, and when the means of dissemination of this disease are considered, show it is not practicable on the average farm to run such experiments and avoid infection.

On the experimental farms, however, results were very satisfactory. Here, thanks to the overseers implicitly carrying out instructions, the results obtained showed that when infection from the untreated area was eliminated the treatment effectively controlled the disease without detriment to the resultant crop.

At Stratford Demonstration Farm, Taranaki, four months after sowing, inspection showed no dry-rot was present on any bulb or leaf of the treated area ($\frac{3}{4}$ acre), but dry-rot was present in the untreated area ($\frac{3}{4}$ acre). Both lots had been sown in contiguous areas, being separated by one drill-width (approximately 4 ft.).

At Gore Experimental Farm, Southland, an inspection made four months after sowing showed that the treated areas were free from dry-rot, whereas in the untreated areas the disease was abundant. Both areas were approximately 1 acre in extent, and were separated by a space of 4 ft. 6 in. Following this inspection a Farmers' Demonstration Day was held, resulting in numerous individuals passing through both areas; consequently when a later inspection was made the disease was found to have been carried over to the treated area.

At Winton Experimental Area, Southland, similar areas of treated and untreated seed were sown (approximately 1 acre of each). Inspection five months after sowing showed dry-rot to be present in "patch" form over the untreated area, but only a single lesion was found on one bulb in the treated area.

One factor brought to light, however, was that in all experimental plots examined there was no apparent difference between treated and untreated areas in so far as the growth of the crop was concerned, demonstrating that, when properly carried out, the treatment does not materially affect germination of the seed.

Although complete control was obtained in the laboratory with the hot Semesan, one-hour soak at 115° F., the use of this method cannot be recommended for general field use, for experience has shown that, unless the seed-germination is high, serious depression of germination may follow treatment.

Consequently a number of lines coming into New Zealand (judging from the 1926 samples treated) would suffer considerably were wholesale treatment carried out. The method is one calling for precision, and therefore cannot be used by the average farmer, so must be undertaken by the Mycological Staff. This, too, presents considerable difficulties, especially with regard to the present method of the sale of various lines; many being forwarded from England in 1 lb. and 2 lb. sealed packages. To open these, treat, and reseal would prove costly. Finally, to treat the swede and turnip seed sold annually in New Zealand (252 tons in 1926, of which 75 tons were swede) would require the

erection of a special plant and the acquisition of a special staff. These factors, combined with certain others of a technical nature, make it appear more feasible to suggest other lines of attack. One such presenting itself is obviously to deal with prevention of infection of the seed before it comes to this country. Another is to grow in New Zealand sufficient swede and turnip seed, dry rot-free, to supply the requirements of the farmer.

To deal with this problem from the former point of view seems more feasible, for the time at least, as then available use can be made of the machinery of distribution, as it were. And such a procedure need not necessarily interfere with the commercial end of seed-distribution. On the other hand, were swede- and turnip-seed-growing in New Zealand successful (and there is nothing to prevent its being a success, under proper organization), competition with commercial firms would be inevitable.

The principal method of seed-production in England is, according to Walker (16) as follows: The seed is sown (presumably in boxes or seed-beds) in August, and transplanted about six weeks later to the main fields, 50,000 plants being planted to the acre, the stock seed being supplied by the seedsmen. The crop remains in the ground throughout the winter and matures the following July or August. The seed-spikes are cut and handled just as is grain, and when dry are stacked in long ricks. A good yield of turnip seed is about 1,800 lb. per acre.

Mother-seed Methods.

Now, it becomes obvious from what has been written that if the seed used for this purpose were disease-free ("mother seed") and sown on land which had not been in cabbages, swedes, or turnips for at least twelve months, a clean crop would result. And it would be a simpler matter to treat the seed used as mother seed than to treat the bulk seed after it was landed in this country. For at most this would necessitate the treatment of about 375 lb. of seed to supply the New Zealand trade. Contrast this with the treatment of 564,480 lb. introduced into this country in 1926!

Should it be possible to ensure clean seed coming into New Zealand, it will be necessary, for the first season at least, for the farmer to observe certain simple precautions. These are (a) to abstain from sowing swedes or turnips on land which was in a similar crop the previous season; (b) to clear absolutely all previous season's seed from his drills, &c.; (c) to destroy with celerity any swede or turnip seed on his property. Merchants will have to prepare the way by ridding their premises of any previous season's swede or turnip seed. This is unnecessary advice to those who handle lines of good seed; but for the small man—the one who buys at a reduced figure from the larger merchant all that seed which the latter was unable to sell the previous season—it becomes necessary if dry-rot is to be eliminated.

The same restrictions must be applied to those engaged in selling garden varieties of swedes and soft turnips, and to those few producing seed of swedes and turnips in New Zealand.

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SUMMARY.

1. Dry-rot was first recorded in New Zealand in 1905; since then it has become the most serious disease of swede and soft turnips.

2. An intensive series of cultural experiments has demonstrated (i) that it may persist for several weeks in mummied bulbs lying on the surface of the ground, a source of infection which can persist only so long as the bulb escapes decay; (ii) that it is carried in the seed.

3. The pathogen is present in the testa (seed-coat). It grows from this to the developing cotyledons, where it produces a small brown lesion in which are formed numerous pycnidia.

4. The spores are embedded in mucilage within the pycnidium. The mucilage is highly hygroscopic, consequently during wet weather it swells and is forced from the pycnidium through an opening provided for the purpose as a long tendril in which the spores are embedded. During wet weather the mucilage is partially dissolved and the spores washed in raindrops to the bulb by way of the channelled surface of the petiole, or carried from an infected to a healthy plant by means of contiguous leaves coming in contact with tendrils, the spores in turn being washed from these leaves to the bulbs.

5. In this manner initial infection centres are established in the field. From these spread is gradual and occurs in a concentric manner, large, somewhat circular infection areas being produced. Finally, one or more of these may merge, when the greater part of the field may show infection.

6. Spread being governed by the amount of moisture present in any one area, it is evident the disease is most severe in low-lying areas and general only where rainfall is abundant. In dry seasons or dry localities it may be slight or absent.

7. When swedes are used for seed purposes the disease spreads from the bulb to the leaves of the flowering-spike, infecting these and the spike; it spreads from this to the developing siliques, forming lesions from which the mycelium spreads to the enclosed seed. Seed infection occurs to the extent of about 1 per cent., but may be greater or less in different lines. In many it may be absent.

8. Inoculation experiments have demonstrated that the lesions produced on bulbs, leaves, spike, siliques are all caused by the same fungus; and that the organisms obtained from seed mostly belong to the same species.

9. The causal organism has been found to consist of several strain-groups, which are characterized by their growth-curves, colour of the mycelium produced on standard media, degree of sporulation, whether staling or non-staling types, whether staining the media or not. Cultures of *Phoma lingam*, *P. Napobrassicae*, *P. Brassicae*, and *P. oleracea*, obtained from abroad, all fall into certain of these strain-groups.

10. Morphological comparisons show that pycnidia of these strain-groups, and pycnidia obtained in cultures of *Phoma lingam*, *P. Napobrassicae*, *P. Brassicae*, and *P. oleracea*, all so closely resemble one another that separation of any one is not possible.

11. Consequently all are considered synonyms of *Phoma lingam*, the prior name applied to those species under consideration.

12. A detailed description of *Phoma lingam* and its strain-groups is given in the text, together with the synonymy of both botanical and common names.

13. *Phoma lingam* has in New Zealand been found on swede, turnip, and cabbage. Hosts elsewhere recorded are also appended following the technical description.

14. A detailed description of the symptoms produced on leaves, stems, siliques, and bulbs is given.

15. Development of the pycnidia and mechanism of spore-discharge is discussed.

16. Laboratory experiments in control have been dealt with fully. The treatment finally adopted—steeping seed for one hour in Semesan 0.25 per cent. solution held at 115° F.—has been shown to give complete control without appreciable damage to the seed, provided the latter is of high germination.

17. This method has been tried out on the field scale, but results were mostly unsatisfactory in that contamination of the treated areas was frequent through the use of cultural implements, &c., though where contamination had been avoided complete control was secured.

18. It is considered that the most efficient method of dealing with the disease is to urge the growers of seed for New Zealand to treat their "mother seed" with the method recommended. In this way the depressing effects of the treatment on seed-germination would prove negligible, necessitating at most the use by the contract grower of a few extra pounds of seed; and, if properly carried out, would ensure that the whole of the seed coming to New Zealand were dry-rot free.

19. Were such seed used, the New Zealand farmer would be required merely (i) to rid his premises and implements from previous season's seed, (ii) to sow clean seed on land which had not been for twelve months previously in Brassicas.

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